Visual-vestibular conflict induced by virtual reality in humans

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Abstract

Conflicting inputs from visual and vestibular afferents produce motion sickness and postural instability. However, the relationship of visual and vestibular inputs to each other remains obscure. In this study, we examined the development of subjective sickness- and balance-related symptoms and objective equilibrium ataxia induced by visual–vestibular conflict (VVC) stimulation using virtual reality. The subjective symptoms evaluated by Graybiel’s and Hamilton’s criteria got gradually worse during the VVC. The objective postural instability was not observed during the VVC, but it occurred immediately after the VVC. There was a time lag between the subjective symptoms and objective ataxia induced by VVC. Our study suggests that the VVC inputs are processed in different pathways causing subjective autonomic symptoms and postural instability in humans.

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Abnormality of spatial orientation provokes motion sickness\cite{11,15} leading to autonomic symptoms and produces postural instability\cite{13}. Previous studies demonstrated that disparity between subjective reports of motion sickness or balance and objective postural instability\cite{1,3,7,14}. However, most studies used ready-made flight simulators, which did not ensure a suitable situation for inducing motion sickness. In these studies, subjective and objective measures were only examined before and after stimulation, because subjects were fixed in the simulator cockpit during the stimulation.

In the present study, we examined the development of subjective symptoms and objective equilibrium ataxia before, during and after visual–vestibular conflict (VVC) stimulation by means of virtual reality (VR). The VR system allowed us to easily coordinate incoherent VVC to induce motion sickness and postural instability. The subjects are free to move their head and body during VR stimulation; thus the assessment of subjective symptoms and objective ataxia during the VVC were easy.

Subjects were nine healthy young volunteers (eight male and one female, mean age: 28.4 years old). The VR system used in the present study was a projection-based system that surrounds the subject with four screens: three rear projection screens for walls and a down-projection screen for a floor space 9 m\textsuperscript{2} each (CAVE; Electronic Visualization Laboratory, University of Illinois, Chicago, IL) (Fig. 1A). Subjects wore liquid crystal stereo shutter glasses to resolve the stereoscopic imaginary. An electromagnetic tracking system attached to the glasses determined the location and angle of user’s head orientation.

Subjects were immersed in two different VR conditions. The background in both conditions was made by a randomized texture pattern (Fig. 1A). One was a Mismatch condition; when subjects turned their head, the background of the VR turned to a degree double that of the head movement on a vertical axis. The other was the Control condition; an interactive computer graphics synchronizing the background image proportionally to the subject motion.

The levels of the severity of motion sickness were given numerical scores according to Graybiel’s criteria: a multi symptom checklist to assess motion sickness symptomatol...
ogy. [2]. Total score ranges from 0 to 50. Higher scores of the questionnaire reflect severer symptoms. The subjective balance symptom questionnaire of Hamilton et al., was also applied, which assesses four balance-related symptoms: walking, balance, visual illusions and leans [1,3]. Total score ranges from 0 to 16. Higher scores of the questionnaire reflect severer symptoms. Body sway was recorded with a force platform (1G06; NEC, Tokyo, Japan) under both conditions with eyes opened and closed. The area covered by the sway path was calculated. Each subject was asked to stand as still as possible on the force platform with both arms beside his body and with his feet close together with eyes open and closed for 60 s each. During measurement with eyes open, subjects were asked to look at a fixation point at eye level 3 m from the front wall. The background was randomized texture pattern desynchronizing (mismatch) or synchronizing (control) background image proportionally to subject motion.

Under mismatch and control conditions, subjects were exposed to 20 min of immersion in the VR and at work on the track of a virtual ball during immersion. This virtual ball task guaranteed the equal quantity of subjects’ locomotion during both the conditions. The virtual ball was regulated to rotate at a velocity of approximately 43.9°/s with the trajectory drawing a randomly distorted clockwise circle about 1 m in diameter at the subject’s eye level. We measured the subjects’ symptoms and postural instability, before the VR immersion (PRE), at the first period of the VR immersion (1st; 1 min after the VR start), at the second period of the VR immersion (2nd; 12 min after the VR start) and immediately after the VR exit (POST) (Fig.1B).

Analysis of variance (two factors of ANOVA with repeated measures) was used for analyzing the scores and postural sway in subjects. The data of four groups (Pre, 1st, 2nd and Post) were compared with post-hoc Scheffé’s test. Difference between mismatch and control conditions’ data was analyzed by paired t-test. The correlation between subjective and objective measures was analyzed by Spearman correlation test.

Under the mismatch condition, the score of Graybiel’s criteria was gradually increased and showed a significantly higher score at the 2nd period of the VVC (Fig. 2A). At the Post period, the score for motion sickness was decreased, but was still significantly higher than that under the control condition. The score for Hamilton’s criteria under the mismatch condition showed the same changes over time as those of Graybiel’s score (Fig. 2B). Under the control condition, the score of both Graybiel’s and Hamilton’s criteria was not significantly changed. Gradual increase in subjective symptoms coincided with the previous report using low frequency horizontal oscillation stimulation [8].

The area of body sway path with both eyes open and closed under the mismatch condition was increased immediately after the VR exit (Post period) and was significantly higher than that under the control condition (Fig. 3), although there were no changes in the area of sway during immersion in the VR (1st and 2nd periods). Because of technical difficulties, the development of objective postural instability during the VVC had not been sufficiently assessed in previous studies. This report is the first to

Fig. 1. Schema of experimental design. (A) Subject in the background visual field created by the VR. (B) Schema of time schedule. Broken line arrows show subjective and objective measures; before the VR immersion (PRE), at the 1st period of the VR immersion (1st; 1 min after the VR start), at the 2nd period of the VR immersion (2nd; 12 min after the VR start) and immediately after the VR exit (POST)

Fig. 2. The severity of the Graybiel’s criteria (A) and Hamilton’s criteria (B) on the immersion in the VR. Closed triangle: the mismatch condition. Closed circle: the control condition. *P < 0.05. Error bar: SE.
examine postural instability during the VVC and indicate a time lag between subjective symptoms (Graybiel’s and Hamilton’s score) and objective ataxia (body sway test with eyes open and eyes closed) due to the VVC.

Large inter-individual variability was observed in subjective and objective measures, which was in agreement with previous findings that vestibular and perceptual responses are not very accurate\[4,5\]. The ranges of individual scores at the 2nd period of the VVC in Hamilton’s questionnaire and Graybiel’s questionnaire were 0.00–5.00 and 0.00–16.00, respectively. The ranges of body path area with eyes open and eyes closed at the Post period of the VVC were 3.50–8.20 and 3.02–6.42, respectively.

The individual Hamilton’s score at any period was not correlated with individual objective body sway. The individual Graybiel’s score at the 2nd period was, however, correlated with individual objective body sway with eyes opened (r = −0.80, P < 0.05). The correlation index of −0.80 indicates that the individual with severer subjective symptoms likely to have lighter objective ataxia, not leading to a meaningful explanation. Graybiel’s score at other periods was not correlated with individual body sway. Taking into account the large inter-individual variability and the reliability of subjective and objective measures, it is not surprising that there is a scarce relation between the two measures. Previous reports also indicated a low relation between subjective reports of motion sickness or balance-related symptoms and postural instability [14].

It has been reported that the nature of the link between motion sickness and postural instability is unclear [6]. According to the sensory conflict theory, incompatible percepts on different sensory channels or a percept incompatible with expectation produces motion sickness and postural instability simultaneously [9,10]. Riccio and Stoffregen hypothesized, however, that prolonged postural instability might be the cause of motion sickness [12]. However, the present study suggests that the preceding symptoms associated with motion sickness are the cause of postural instability.

It has been reported that the interaction between spatial orientation perception and reflexive eye movements are also contradictory [4,5]. In darkness, neither nystagmus nor sensation can be used as a valid index of the other. Ivanenko et al. [5] suggested that integration of semicircular canal output, allowing compensatory eye movements and subjective head orientation, might occur through different pathways. Similarly, the present results strongly suggest that VVC inputs are processed in different pathways causing subjective symptoms and postural instability.

In conclusion, the incoherent VVC induced by VR was sufficient to induce motion sickness and postural instability. The present study firstly revealed the time lag between subjective symptoms of motion sickness and objective postural instability. The autonomic symptoms and postural equilibrium induced by VVC may occur through different pathways.

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